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SEARCH REQUEST FORM

Scientific and Technical Information Center

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Requester's Full Name: Natalie Dawson Examiner #.: 78462 Date: 4-23-01
Art Unit: 1642 Phone Number 30 8-6410 Serial Number: 09/721183
Mail Box and Bldg/Room Location: 8E12 CM1 4C01 Results Format Preferred (circle): PAPER DISK E-MAIL

If more than one search is submitted, please prioritize searches in order of need.

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Please provide a detailed statement of the search topic, and describe as specifically as possible the subject matter to be searched. Include the elected species or structures, keywords, synonyms, acronyms, and registry numbers, and combine with the concept or utility of the invention. Define any terms that may have a special meaning. Give examples or relevant citations, authors, etc, if known. Please attach a copy of the cover sheet, pertinent claims, and abstract.

Title of Invention: _____

Inventors (please provide full names): _____

Earliest Priority Filing Date: 11-23-99

***For Sequence Searches Only* Please include all pertinent information (parent, child, divisional, or issued patent numbers) along with the appropriate serial number.**

Please search BCSG as it relates to ~~[redacted]~~ breast cancer + metastases. Also for use in identifying therapeutic agent that may be used in treating breast cancer.

RECEIVED APR 26 2001 (STIC)

Point of Contact:
Beverly Shears
Technical Info. Specialist
CM1 12C14 Tel: 308-4884

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STAFF USE ONLY | **Type of Search** | **Vendors and cost where applicable**

Searcher: Beverly 4-27-01 | NA Sequence (#) _____ | STN _____

Searcher Location:		Structure (#)		Questel/Orbit	
Date Searcher Picked Up:		Bibliographic		Dr.Link	
Date Completed:	04-26-01	Litigation		Lexis/Nexis	
Searcher Prep & Review Time:	10	Fulltext		Sequence Systems	
Clerical Prep Time:		Patent Family		WWW/Internet	
Online Time:	21	Other		Other (specify)	

PTO-1590 (1-2000)

FILE 'CAPLUS' ENTERED AT 11:54:40 ON 26 APR 2001
L1 3 S BCSG
L2 30 S CANCER SPECIF? GENE
L3 9 S L2 AND (BREAST OR MAMMAR?)
L4 12 S L1 OR L3

L4 ANSWER 1 OF 12 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 2000:421413 CAPLUS
DOCUMENT NUMBER: 133:54509
TITLE: Methods of identifying genes differentially expressed in primary **breast** cancer cells selected by monoclonal antibody to fibroblast activation protein (FAP)
INVENTOR(S): Mackay, Alan Gordon; O'Hare, Michael John
PATENT ASSIGNEE(S): Ludwig Institute for Cancer Research, USA
SOURCE: PCT Int. Appl., 70 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000036420	A2	20000622	WO 1999-GB4183	19991210
WO 2000036420	A3	20001019		

W: CN, JP, US
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE

PRIORITY APPLN. INFO.: GB 1998-27430 A 19981211
AB A differential display PCR method for identifying genes that are differentially expressed in tumor cells, preferably primary **breast** tumor cells selected by monoclonal antibody to fibroblast activation protein (FAP) is described. FAP is a cell-surface glycoprotein mainly expressed in stromal fibroblasts of epithelial cancers. The monoclonal antibody F19 to FAP is used to select primary **breast** cancer cells from solid **breast** tumor samples, which are used to identify tumor specific candidate marker genes. RT-PCR is performed from mRNA of selected tumor cells and control cells (normal **breast** organoids and normal human tissues), and 29 unique bands from selected tumor cells are isolated and sequenced. All these clones are compared to the database and one of them is further extended for full length sequences by cDNA library screening and RACE PCR. These **breast cancer specific genes** can be used in cancer diagnosis, treatments, and drug screening.

L4 ANSWER 2 OF 12 CAPLUS COPYRIGHT 2001 ACS

ACCESSION NUMBER: 2000:209944 CAPLUS
DOCUMENT NUMBER: 132:248258
TITLE: Method of diagnosing, monitoring, staging, imaging and treating gynecologic cancers and testicular cancer
INVENTOR(S): Ali, Shujath M.; Cafferkey, Robert
PATENT ASSIGNEE(S): Diadexus Llc, USA
SOURCE: PCT Int. Appl., 36 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000016805	A1	20000330	WO 1999-US21774	19990923

W: CA, JP, US
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE
PRIORITY APPLN. INFO.: US 1998-101522 P 19980923
AB The present invention provides a new method for detecting, diagnosing, monitoring, staging, prognosticating, imaging and treating gynecol. cancers and testicular cancer.
REFERENCE COUNT: 3
REFERENCE(S): (1) Amrad Operations Pty Ltd; WO 9836054 1998 CAPLUS
(2) Inuoe, M; Biochemical and Biophysical Research Communications 1998, V252, P307
(3) Ricke, D; Homo sapiens chromosome 16, cosmid clone 352F10 (LANL) 1998

L4 ANSWER 3 OF 12 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 2000:161492 CAPLUS
DOCUMENT NUMBER: 132:204018
TITLE: Diagnosis and staging of various cancers by detection of **cancer-specific genes** (CSG) and antibody-based treatment
INVENTOR(S): Salceda, Susana; Sun, Yongming; Recipon, Herve; Cafferkey, Robert
PATENT ASSIGNEE(S): Diadexus Llc, USA
SOURCE: PCT Int. Appl., 58 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000012758	A1	20000309	WO 1999-US19655	19990901

W: CA, JP, US
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE

PRIORITY APPLN. INFO.: US 1998-98880 19980902

AB The present invention provides a new method for detecting, diagnosing, monitoring, staging, and prognosticating selected cancers including gynecol. cancers such as **breast**, ovarian, uterine and endometrial cancer and lung cancer by measurement of the levels of **cancer-specific genes** (CSG) in cells, tissue, or bodily fluid of a control patient and in a cancer patient, where elevated CSG levels indicated the presence of cancer, and further elevated levels the occurrence of metastasis. **Cancer-specific gene** sequences are presented which may be used as diagnostic markers for the presence of CSG. Antibodies to these sequences labeled with paramagnetic ions or radioisotopes may be used for imaging the cancer, and antibodies cojugated to cytotoxic agents may be used therapeutically.

REFERENCE COUNT: 3
REFERENCE(S): (1) Croce; US 5939258 A 1999 CAPLUS
(2) Paoloni-Giacobno; Genomics 1997, V44, P309
(3) Yu; US 5733748 A 1998 CAPLUS

L4 ANSWER 4 OF 12 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1999:692247 CAPLUS
DOCUMENT NUMBER: 132:18563
TITLE: Search for novel proteins involved in the development of chemoresistance in colorectal cancer and fibrosarcoma cells in vitro using two-dimensional electrophoresis, mass spectrometry and microsequencing
AUTHOR(S): Sinha, Pranav; Hutter, Gero; Kottgen, Eckart; Dietel, Manfred; Schadendorf, Dirk; Lage, Hermann
CORPORATE SOURCE: Institut fur Laboratoriums-medizin und Pathobiochemie, Universitatsklinikum Charite, Berlin, Germany
SOURCE: Electrophoresis (1999), 20(14), 2961-2969
CODEN: ELCTDN; ISSN: 0173-0835
PUBLISHER: Wiley-VCH Verlag GmbH
DOCUMENT TYPE: Journal
LANGUAGE: English

AB In search of novel mechanisms that may lead to the development of chemoresistance of malignant tumors of the large bowel, we used two-dimensional electrophoresis to identify proteins that were

overexpressed in colorectal and fibrosarcoma cell lines that were resistant towards mitoxantrone. This cytostatic drug is known to lead to atypical multidrug resistance, i.e., the classical mechanism of multidrug resistance (MDR) accompanied by the overexpression of P-glycoprotein (P-gp) is ineffective. Using mass spectrometry and microsequencing we found adenine phosphoribosyl transferase and **breast cancer specific gene** 1 (BCSG1) overexpressed in the resistant colorectal tumor cell line. In the chemoresistant fibrosarcoma cell line we found two proteins that were overexpressed. One was identified as Rho-guanine dinucleotide phosphate (Rho-GDP) dissocn. inhibitor and the other had sequence homologies with yeast protein yer-7. The putative role of these proteins is discussed.

REFERENCE COUNT: 42
REFERENCE(S):
(1) Altland, K; Clin Chem 1984, V30, P2098 CAPLUS
(2) Antonarakis, S; Nature Genet 1998, V19, P106 CAPLUS
(3) Barger, S; Biochem J 1995, V311, P45 CAPLUS
(4) Cheung, C; Ann Clin Biochem 1987, V24, P140 CAPLUS
(6) Coso, O; Cell 1995, V81, P1137 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 5 OF 12 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1999:495387 CAPLUS
DOCUMENT NUMBER: 131:154486
TITLE: Human genes and gene expression products from a colon cancer cell line KM12L4-A cDNA library
INVENTOR(S): Williams, Lewis T.; Escobedo, Jaime; Innis, Michael A.; Garcia, Pablo Dominguez; Sudduth-Klinger, Julie; Reinhard, Christoph; Giese, Klause; Randazzo, Filippo; Kennedy, Giulia C.; Pot, David; Kassam, Altaf; Lamson, George; Drmanac, Radoje; Crkvenjakov, Radomir; Dickson, Mark; Drmanac, Snezana; Labat, Ivan; Leshkowitz, Dena; Kita, David; Garcia, Veronica; Jones, William Lee; Stache-Crain, Birjit
PATENT ASSIGNEE(S): Chiron Corporation, USA; Hyseq Inc.
SOURCE: PCT Int. Appl., 2479 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

WO 9938972 A2 19990805 WO 1999-US1619 19990128
WO 9938972 A3 19991223
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
AU 9924716 A1 19990816 AU 1999-24716 19990128
EP 1053319 A2 20001122 EP 1999-904288 19990128
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI

PRIORITY APPLN. INFO.:

US 1998-72910	P	19980128
US 1998-75954	P	19980224
US 1998-80114	P	19980331
US 1998-80515	P	19980403
US 1998-80666	P	19980403
US 1998-105234	P	19981021
US 1998-105877	P	19981027
WO 1999-US1619	W	19990128

AB This invention relates to novel human polynucleotides and variants thereof, their encoded polypeptides and variants thereof, to genes corresponding to these polynucleotides and to proteins expressed by the genes. The invention provides the nucleotide sequences for 2502 human polynucleotides isolated as cDNA clones from the human colon cancer cell line KM12L4-A, 2600 validation sequence, plus 146 sequences assembled as contigs. Many of the cDNA sequences provided are differentially expressed in the cancerous state (colon cancer, lung cancer, breast cancer) or in specific tissues (e.g., colon). Database homol. searches identified various protein families that encompass some of the putative protein products. Diagnostic and therapeutic agents employing such novel human polynucleotides, their corresponding genes or gene products, e.g., these genes and proteins, including probes, antisense constructs, and antibodies, are also provided.

L4 ANSWER 6 OF 12 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1999:110205 CAPLUS
DOCUMENT NUMBER: 130:265716
TITLE: Stimulation of **breast** cancer invasion and metastasis by synuclein .gamma.
AUTHOR(S): Jia, Tongli; Liu, Yiliang E.; Liu, Jingwen; Shi, Y. Eric
CORPORATE SOURCE: Departments of Pediatrics [T. J., Y. E. L. Y. E. S] and Pathology, Long Island Jewish Medical

Center, The Long Island Campus for the Albert Einstein College of Medicine, New Hyde Park, NY, 11040, USA
SOURCE: Cancer Res. (1999), 59(3), 742-747
CODEN: CNREA8; ISSN: 0008-5472
PUBLISHER: AACR Subscription Office
DOCUMENT TYPE: Journal
LANGUAGE: English
AB We recently identified and cloned novel **breast cancer-specific gene** BCSGI by direct differential cDNA sequencing. BCSGI has a great sequence homol. with the Alzheimer's disease-related neural protein synuclein (SNC); thus, it was also named SNC-.gamma.. Overexpression of SNC-.gamma. in **breast** cancer cells leads to a significant increase in motility and invasiveness in vitro and a profound augmentation of metastasis in vivo. Our data suggest that this member of the neural protein SNCs might have important functions outside the central nervous system and may play a role in **breast** cancer progression.
REFERENCE COUNT: 28
REFERENCE(S):
(2) Clayton, D; Trends Neurosci 1998, V21, P249 CAPLUS
(3) Deb, S; J Neurochem 1996, V66, P1641 CAPLUS
(4) Douglas, A; Int J Cancer 1998, V75, P64 CAPLUS
(6) Jakes, R; FEBS Lett 1994, V345, P27 CAPLUS
(7) Ji, H; Cancer Res 1997, V57, P759 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 7 OF 12 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1998:798284 CAPLUS
DOCUMENT NUMBER: 130:192430
TITLE: Deciphering molecular circuitry using high-density DNA arrays
AUTHOR(S): Mack, David H.; Tom, Edward Y.; Mahadev, Mamatha; Dong, Helin; Mittmann, Michael; Dee, Suzanne; Levine, Arnold J.; Gingeras, Thomas R.; Lockhart, David J.
CORPORATE SOURCE: Program in Cancer Biology, Santa Clara, CA, 95051, USA
SOURCE: Pezcoller Found. Symp. (1998), 9(Biology of Tumors), 85-108
CODEN: PFSYES; ISSN: 0961-785X
PUBLISHER: Plenum Publishing Corp.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB DNA arrays contg. oligonucleotides complementary to > 6,500 human EST's were used to generate normal and **breast**

cancer specific gene expression profiles. More than 1,500 expressed genes were detected in both cell types. Over 300 genes demonstrated significantly different levels of expression between normal and transformed cells. Increased mRNA levels were obsd. for the Her2/neu oncogene and genes involved in tis signal transduction, including Grb-7, Ras, Raf, Mek, and ERK. In addn., a simple categorization of the expression changes revealed patterns characteristic of loss of wild-type p53 function. Genotyping of the p53 locus using a DNA resequencing array reveled inactivating mutation in the p53 DNA-binding domain and loss of heterogeneity. These data demonstrate a general array-hybridization approach to deciphering biochem. pathways and generating testable hypotheses concerning the mechanisms of cell growth and differentiation.

REFERENCE COUNT: 32
REFERENCE(S):
(1) Adams, M; Nature 1995, V377, P3 CAPLUS
(3) Boguski, M; Nature Genetics 1993, V4, P332 CAPLUS
(8) Fodor, S; Science 1991, V251, P767 CAPLUS
(21) Schena, M; Science 1995, V270, P467 CAPLUS
(22) Sivaraman, V; J Clin Invest 1997, V99(7), P1478 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 8 OF 12 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1998:543170 CAPLUS
DOCUMENT NUMBER: 129:171517
TITLE: **Breast cancer specific gene** 1
INVENTOR(S): Ji, Hongjun; Rosen, Craig A.
PATENT ASSIGNEE(S): Human Genome Sciences, Inc., USA
SOURCE: PCT Int. Appl., 73 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9833915	A1	19980806	WO 1998-US1804	19980203

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES,

FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

AU 9862572	A1	19980825	AU 1998-62572	19980203
EP 1015582	A1	20000705	EP 1998-904776	19980203

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI

PRIORITY APPLN. INFO.:	US 1997-37080	P	19970203
	WO 1998-US1804	W	19980203

AB CDNA sequences are provided encoding the human **Breast cancer specific gene** 1 [BCSG1] protein. BCSG1 polypeptides are also provided as are vectors, host cells and recombinant methods for producing the same. Lastly, diagnostic methods for detecting **breast** cancer are described.

L4 ANSWER 9 OF 12 CAPLUS COPYRIGHT 2001 ACS

ACCESSION NUMBER:	1997:128808 CAPLUS
DOCUMENT NUMBER:	126:221387
TITLE:	Identification of a **breast cancer-specific gene**, BCSG1, by direct differential cDNA sequencing
AUTHOR(S):	Ji, Hongjun; Liu, Yiliang E.; Jia, Tongli; Wang, Mingsheng; Liu, Jingwen; Xiao, Guowei; Joseph, Benjamin K.; Rosen, Craig; Shi, Y. Eric
CORPORATE SOURCE:	Dep. of Pediatrics, Long Island Jewish Med. Center, Rocksville, MD, 20850-3338, USA
SOURCE:	Cancer Res. (1997), 57(4), 759-764 CODEN: CNREA8; ISSN: 0008-5472
PUBLISHER:	American Association for Cancer Research
DOCUMENT TYPE:	Journal
LANGUAGE:	English

AB A high-throughput direct-differential cDNA sequencing approach was employed to identify genes differentially expressed in normal **breast** as compared with **breast** cancer. Approx. 6000 expressed sequence tags (ESTs) from cDNA libraries of normal **breast** and **breast** carcinoma were selected randomly and subjected to EST-sequencing anal. The relative expression levels of more than 2000 unique EST groups were quant. compared in normal vs. cancerous **breast**. Of many putative differentially expressed genes, a **breast cancer-specific gene**, BCSG1, which was expressed in high abundance in a **breast** cancer cDNA library but scarcely in a normal **breast** cDNA library, was identified as a putative **breast** cancer marker. In situ hybridization anal. demonstrated stage-specific BCSG1 expression as follows: BCSG1 was undetectable in normal or benign **breast** lesions, showed partial expression in ductal carcinoma in situ, but was expressed at an extremely high level in advanced infiltrating **breast** cancer. The predicted amino acid sequence of BCSG1 gene has a

significant sequence homol. to the non-amyloid .beta. protein fragment of the Alzheimer's disease amyloid protein. BCSG1 overexpression may indicate **breast** cancer malignant progression from benign **breast** or in situ carcinoma to the highly infiltrating carcinoma.

L4 ANSWER 10 OF 12 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1982:66141 CAPLUS
DOCUMENT NUMBER: 96:66141
TITLE: Glycopeptides prepared from mouse cerebrum inhibit protein synthesis and cell division in baby hamster kidney cells, but not in their polyoma virus-transformed analogs
AUTHOR(S): Kinders, Robert J.; Johnson, Terry C.
CORPORATE SOURCE: Div. Biol., Kansas State Univ., Manhattan, KS, 66506, USA
SOURCE: Exp. Cell Res. (1981), 136(1), 31-41
CODEN: ECREAL; ISSN: 0014-4827
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Glycopeptides isolated from mouse cerebral cortex cell surfaces (**BCSG**) inhibited cell growth and protein synthesis in baby hamster kidney (BHK)-21 cells, whereas polyoma vs.-transformed BHK-21 cells (pyBHK-21) were refractory to the inhibitory activity of the glycopeptides. Growth inhibition was reversible and nonlethal to BHK-21 cells. Despite that difference in sensitivity to the action of the glycopeptides, both cell lines could bind the inhibitor in a saturable fashion and in similar quantities. After trypsinization, BHK-21 cells appeared refractory to the inhibitor, whereas pyBHK-21 cells became sensitive, suggesting the presence of a receptor for **BCSG** on the cell surface of both cell lines. Incubating **BCSG** with conditioned medium from pyBHK-21 cells resulted in loss of the glycopeptide's inhibitory activity. In contrast, medium conditioned by BHK-21 cells had no effect on the inhibitory activity of **BCSG**. The refractoriness of pyBHK-21 cells to **BCSG** may be related to their autonomous growth characteristics and failure to respond to topo-inhibitory growth control. **BCSG** may be a naturally occurring growth regulator whose function can be explored by use of the BHK-21/pyBHK-21 model system.

L4 ANSWER 11 OF 12 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1981:566980 CAPLUS
DOCUMENT NUMBER: 95:166980
TITLE: A cell surface glycoprotein virus inhibitor that is not interferon
AUTHOR(S): Johnson, Terry C.; Kinders, Robert J.; McGee, James E.

CORPORATE SOURCE: Div. Biol., Kansas State Univ., Manhattan, KS, 66506, USA
SOURCE: Biochem. Biophys. Res. Commun. (1981), 102(1), 328-34
CODEN: BBRCA9; ISSN: 0006-291X
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The possible relationship between a newly isolated glycoprotein virus inhibitor and interferon was assessed. Comparisons of the cell surface glycopeptide, obtained from mouse cerebral cortex, and interferon included antiviral activity, radioimmune assays, and the ability of antibodies raised against the brain cell surface glycoprotein (**BCSG**) and against mouse L cell interferon to ppt. the biol. activity. **BCSG** was able to inhibit virus replication but only in a transient fashion. Although anti-**BCSG** pptd. a major portion of the radiolabeled inhibitor in a double antibody assay, anti-mouse interferon did not. Over 90% of the inhibitory activity was removed with anti-**BCSG** and Staphylococcus protein A while anti-mouse interferon removed little, or none, of the activity under similar reaction conditions. Other properties of the **BCSG** that distinguish it from interferon are presented.

L4 ANSWER 12 OF 12 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1980:492476 CAPLUS
DOCUMENT NUMBER: 93:92476
TITLE: Glycopeptides from brain inhibit rates of polypeptide chain elongation
AUTHOR(S): Kinders, Robert J.; Hughes, Joseph V.; Johnson, Terry C.
CORPORATE SOURCE: Div. Biol., Kansas State Univ., Manhattan, KS, 66506, USA
SOURCE: J. Biol. Chem. (1980), 255(13), 6368-72
CODEN: JBCHA3; ISSN: 0021-9258
DOCUMENT TYPE: Journal
LANGUAGE: English

AB When baby hamster kidney (BHK)-21 cells were infected with vesicular stomatitis virus (a neg. strand RNA virus) cell-surface glycopeptides from mouse cerebrum (**BCSG**) extensively inhibited viral protein synthesis. This inhibition was effective against both protein and glycoprotein synthesis and was independent of amino acid uptake by infected cells, synthesis of viral RNA, and degrdn. of viral proteins. Anal. of polyribosome profiles in uninfected BHK-21 cells indicated that the degree of cellular protein synthesis inhibition could not be attributed to activation of RNase or solely to a disruption of chain initiation. When added directly to a cell-free protein-synthesizing system derived from BHK-21 cells, **BCSG** was ineffective, but if the inhibitory

material was first allowed to react with cells, cell-free protein synthesis was substantially reduced. When **BCSG** were reacted with cells for 5 min at 0.degree., BHK-21 (a **BCSG** -sensitive line) and murine fibrosarcoma 2237 (a **BCSG** -insensitive line) both effectively adsorbed the inhibitor from the medium.

(FILE 'CAPLUS' ENTERED AT 11:54:40 ON 26 APR 2001)
L5 3 S CSG AND (BREAST OR MAMMAR?)
L6 2 S L5 NOT L4

L6 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 2001:196797 CAPLUS
TITLE: Establishment of La-tPA/G-CSF dual transgenic mice and expression in their **mammary** gland
AUTHOR(S): Lu, Yifan; Tian, Chai; Deng, Jixian; Cheng, Xuan; Huang, Peitang
CORPORATE SOURCE: Institute of Biotechnology, Academy of Military Medicine Science, Beijing, 100071, Peop. Rep. China
SOURCE: Sci. China, Ser. C: Life Sci. (1999), 42(3), 330-336
CODEN: SCCLFO; ISSN: 1006-9305
PUBLISHER: Science in China Press
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Expression vectors of human granulocyte colony stimulating factor (G-**CSG**) and long acting tissue plasminogen activator (La-tPA) in **mammary** gland were constructed using promoters of mouse whey acid protein gene (WAP) and sheep .beta.-lactoglobulin gene (BLG) with sizes of 2.6 and 5 kb resp. Two kinds of transgenic mice of G-CSF and La-tPA were produced with microinjection. The expression of G-CSF and La-tPA was achieved in **mammary** glands of transgenic mice, resp. In order to establish dual transgenic mice of La-tPA /G-CSF, transgenic mice carrying G-CSF and La-tPA gene characterized with specific expression in **mammary** gland were mated. La-tPA/G-CSF dual transgenic mice were screened out from the hybrid offspring by Once-PCR. The co-expression of La-tPA and G-CSF in **mammary** gland of the dual transgenic mice was confirmed by the milk assayed and Northern blot anal. Some parameters about the dual transgenic mice indicated that there were fewer litters than that of normal mice. The ratio of dual transgenes was 46.1% in F1 generation, and offspring's sex ratio was normal. Hence a dual transgenic mouse model was established for the study of co-expression foreign proteins in **mammary** gland.
REFERENCE COUNT: 13

REFERENCE(S): (1) Bonifer, C; J Cell Biochem 1991, V47, P99 CAPLUS
(2) Carver, A; Bio/Technology 1993, V11, P1263 CAPLUS
(4) Clark, A; Bio/Technology 1992, V10, P1450 CAPLUS
(5) Efrat, S; Proc Natl Acad Sci USA 1994, V91, P2051 CAPLUS
(6) Gordon, K; Bio/Technology 1987, V5, P1183 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1999:298090 CAPLUS
DOCUMENT NUMBER: 131:111015
TITLE: Extract of Solanum muricatum (pepino/**CSG**) inhibits tumor growth by inducing apoptosis
AUTHOR(S): Ren, Weiping; Tang, Dean G.
CORPORATE SOURCE: Virotech Canada Inc., Windsor, ON, N8W 3K5, Can.
SOURCE: Anticancer Res. (1999), 19(1A), 403-408
CODEN: ANTRD4; ISSN: 0250-7005
PUBLISHER: International Institute of Anticancer Research
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Apoptosis, or programmed cell death, is characterized by certain distinct morphol. and biochem. features. Most chemotherapeutic drugs exert their anti-tumor effects by inducing apoptosis. Therefore, an effective compd. inducing apoptosis appears to be a relevant strategy to suppress various human tumors. In a search for tumor inhibitors from various kinds of plants, we found that exts. from Solanum muricatum (**CSG**) can inhibit tumor growth both in vivo and in vitro by inducing apoptosis. A lyophilized aq. fraction extd. from Solanum muricatum (**CSG**) was used in this study. The human cell lines tested include: prostate (PC3, DU145), stomach (MKN45), liver (QGY-7721, SK-HEP-1), **breast** (MDA-MB-435), ovarian (OVCAR), colon (HT29) and lung (NCI-H209) cancer cells; NHP (prostate), HUVEC (umbilical vein endothelial cell), and WI-38 (lung diploid fibroblasts) normal cells. The cell survival was detd. by either Cell Titer MTS cell proliferation kit or trypan blue dye exclusion assay. The apoptosis was analyzed by (a) apoptotic morphol. by light microscopy; (b) DNA ladder formation; (c) PARP cleavage assay. A) **CSG** possesses selective cytotoxic activity against all the tumor cell lines being tested. The LD50 value is 561-825 .mu.g/mL. B) **CSG** showed a much lower cytotoxicity to NHP, HUVEC and WI-38 normal cell lines with LD50 value being 2.8-3.2 mg/mL, which is 3-6 fold higher than on tumor cells. C) The in vivo study demonstrated that injection of **CSG** (100 .mu.g) directly into tumor mass can

reduce the tumor vol. dramatically in nude mice inoculated with MKN45 gastric cancer cells. D) **CSG**-mediated tumor growth inhibition is through induction of apoptotic cell death, as manifested by (a) typical apoptotic morphol.; (b) DNA ladder formation; and (c) PARP cleavage assay. Taken together, the present study suggests, for the first time, that **CSG** may represent promising new chem. entity which preferentially targets various tumor cells by triggering apoptosis.

REFERENCE COUNT: 17
REFERENCE(S):
(2) Chiang, H; Anticancer Res 1991, V11, P1911 CAPLUS
(4) Hickman, J; Cancer Metastasis Rev 1992, V11, P121 CAPLUS
(5) Hsu, S; Biochem Biophys Res Com 1996, V229, P1 CAPLUS
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ALL CITATIONS AVAILABLE IN THE RE FORMAT

(FILE 'MEDLINE, BIOSIS, EMBASE, WPIDS, CONFSCI, SCISEARCH, JICST-EPLUS, JAPIO, CANCERLIT' ENTERED AT 11:57:13 ON 26 APR 2001)

L7 37 SEA ABB=ON PLU=ON L1 OR L3 OR ((CSG(S) CANCER(W) SPECIF?) AND (BREAST OR MAMMAR?))
L8 17 DUP REM L7 (20 DUPLICATES REMOVED)

L8 ANSWER 1 OF 17 WPIDS COPYRIGHT 2001 DERWENT INFORMATION LTD
ACCESSION NUMBER: 2000-256657 [22] WPIDS
DOC. NO. CPI: C2000-078328
TITLE: Diagnosing, staging, monitoring, imaging and treating cancer especially gynecological cancers e.g. **breast**, ovarian cancer and lung cancer, involves measuring **cancer specific gene** levels in cells and body fluids.
DERWENT CLASS: B04 D16
INVENTOR(S): CAFFERKEY, R; RECIPON, H; SALCEDA, S; SUN, Y
PATENT ASSIGNEE(S): (DIAD-N) DIADEXUS LLC
COUNTRY COUNT: 21
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2000012758	A1	20000309	(200022)*	EN	58

RW: AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE
W: CA JP US

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000012758 A1		WO 1999-US19655	19990901

PRIORITY APPLN. INFO: US 1998-98880 19980902
AN 2000-256657 [22] WPIDS
AB WO 200012758 A UPAB: 20000508
NOVELTY - Detecting, diagnosing metastasis and staging cancer by measuring levels of **cancer specific genes** (**CSG**) in cells, tissues or body fluids, is new. Their remission and progression, decreases and increases in **CSG** levels, is also monitored, by periodic sample analysis.
DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for an antibody (I) against CSG which comprises a 2587, 576, 2070, 890, 1709, 406, 2479, 462 or 272 base pair sequence, all fully defined in the specification.
ACTIVITY - Cytostatic. No supporting data given.
MECHANISM OF ACTION - None given.
USE - The methods are useful for detecting, diagnosing, monitoring, staging, prognosing cancers, especially gynecologic cancers which include ovarian, **breast**, endometrial and uterine cancer (claimed) and lung cancer. (I) labeled with paramagnetic ions or a radioisotope is useful for imaging cancer and (I) conjugated with a cytotoxic agent is useful for treating cancer (claimed).
ADVANTAGE - The discrimination between metastasized and non-metastasized cancers, which was not possible using prior techniques, can be achieved using this method.
Dwg.0/0

L8 ANSWER 2 OF 17 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 2000:299378 BIOSIS
DOCUMENT NUMBER: PREV200000299378
TITLE: Transcriptionally regulated adenoviruses for prostate-specific gene therapy.
AUTHOR(S): Lu, Yi; Steiner, Mitchell S.
SOURCE: World Journal of Urology, (April, 2000) Vol. 18, No. 2, pp. 93-101. print.
ISSN: 0724-4983.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Most virally based vectors for gene therapy contain viral promoters that are tissue-nonspecific. Consequently, unintended expression of toxic therapeutic genes in normal tissues may potentially occur. We

have constructed adenoviruses that contain a bacterial beta-galactosidase (beta-gal) gene (lacZ) under the control of three different prostate-specific promoters: prostate-specific antigen (PSA), probasin, and the mouse **mammary**-tumor-virus long terminal repeat (MMTV; prostate-specific Ad-lacZ). In general, these prostate-specific Ad-lacZ can effectively transduce and express beta-gal in prostate cells and display weak, if any, expression of beta-gal in nonprostate cells in vitro. In vivo, these adenoviruses showed a high level of beta-gal expression in canine prostate but also disseminated to tissues other than prostate after intraprostatic (i.p.) injection. However, none of the prostate-specific Ad-lacZ expressed beta-gal in these nonprostate tissues. Furthermore, prostate-specific Ad-lacZ expressed beta-gal only in xenograft tumors grown in nude mice, derived from human prostate-cancer cells DU145 and PPC-1, but showed no beta-gal expression in tumors derived from human bladder-cancer cells RT4. These results indicate that adenoviruses containing prostate-specific promoters may express intended transgenes specifically in prostate in vivo.

L8 ANSWER 3 OF 17 WPIDS COPYRIGHT 2001 DERWENT INFORMATION LTD
ACCESSION NUMBER: 2000-116557 [10] WPIDS
DOC. NO. CPI: C2000-035637
TITLE: Novel RNA biomarkers for diagnosis, prognosis and management of prostate, **breast** and bladder cancer.
DERWENT CLASS: B04 D16
INVENTOR(S): AN, G; O'HARA, S M; RALPH, D; VELTRI, R W
PATENT ASSIGNEE(S): (UROC-N) UROCOR INC
COUNTRY COUNT: 22
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9964631	A1	19991216	(200010)*	EN	191
RW: AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE					
W: AU CA JP					
AU 9945604	A	19991230	(200022)		
EP 1086246	A1	20010328	(200118)	EN	
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE					

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 9964631	A1	WO 1999-US13151	19990611
AU 9945604	A	AU 1999-45604	19990611
EP 1086246	A1	EP 1999-928561	19990611

WO 1999-US13151 19990611

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9945604	A	Based on	WO 9964631
EP 1086246	A1	Based on	WO 9964631

PRIORITY APPLN. INFO: US 1998-97199 19980612

AN 2000-116557 [10] WPIDS

AB WO 9964631 A UPAB: 20000228

NOVELTY - Nucleic acid markers of prostate, **breast** and bladder cancer are new. The markers are indicators of malignant transformation of prostate, **breast** and bladder tissues and are diagnostic of the potential for metastatic spread of malignant prostate tumors.

DETAILED DESCRIPTION - An isolated nucleic acid segment comprising a full length sequence or the full length complement of a sequence selected from the sequences (A)-(T) of 391, 614, 757, 673, 358, 166, 107, 183, 92, 174, 132, 135, 471, 209, 407, 267, 333, 369, 301 and 25 bp, respectively (T) or 135 amino acid (aa) sequence (U) (sic) (all sequences are given in the specification).

INDEPENDENT CLAIMS are also included for the following:

(1) an isolated nucleic acid molecule (NAM), of 14-100 bp, identical in sequence to a contiguous portion of at least 14 bp of (A)-(U);

(2) an isolated polypeptide with an aa sequence encoded by (A)-(S);

(3) an isolated peptide of 10-050 aa with an aa sequence encoded by (A)-(S);

(4) identifying markers for human prostate cancer by:

(a) providing human prostate RNAs;

(b) amplifying the RNAs;

(c) separating the amplification products;

(d) identifying RNAs that are differentially expressed between human prostate cancers versus normal/benign human prostate;

(5) detecting prostate cancer cells in biological sample by detecting a prostate cancer marker which is a polynucleotide selected from (A)-(U), and the 610 bp (V), 1649 bp (W) or 175 bp (X) sequences given in the specification;

(6) treating prostate cancer by:

(a) obtaining a prostate cancer tissue sample;

(b) screening the sample for expression of a polypeptide encoded by (A)-(U);

(c) providing an antibody that reacts immunologically against the polypeptide, and

(d) administering the antibody;

(7) treating prostate cancer by:

(a) as in (6a) and (6b);

(b) providing an antisense DNA molecule encoding a RNA molecule that binds to the polynucleotide;

(c) providing the antisense DNA molecule in the form of a human vector containing regulatory elements for the production of the RNA molecule; and

(d) administering the vector;

(8) a kit for detecting prostate cancer cells in a biological sample, comprising a primer pair for amplifying (A)-(U), and containers for each of the primers;

(9) a kit for detecting prostate cancer cells comprising a primer pair for amplifying a nucleic acid product of a human gene selected from cyclin A, fibronectin, and a truncated form of Her2/neu, and containers for each of the primers;

(10) a kit for detecting prostate cancer cells comprising an oligonucleotide probe (ON) which binds to a sequence (A)-(U), and a container for the probe;

(11) a kit for detecting prostate cancer cells comprising ON probe binding to a nucleic acid product of a human gene as in (9), and a container for the probe;

(12) a kit for detecting prostate cancer cells comprising an antibody which binds to a protein encoded by (A)-(U), and a container for the antibody;

(13) detecting prostate cancer cells comprising:

(a) providing an antibody binding as in (12) and to a truncated form of Her2/neu;

(b) contacting a human tissue sample with the antibody;

(c) separating bound from unbound antibody; and

(d) detecting the bound antibody;

(14) a kit for detecting prostate cancer cells comprising an antibody binding as in (13), and container for the antibody;

(15) treating prostate cancer by:

(a) selecting a prostate cancer marker selected from (A)- (U), cyclin A, fibronectin and a truncated form of Her2/neu;

(b) providing an inhibitor designed to bind specifically to the protein product of the marker; and

(c) administering the inhibitor;

(16) an isolated nucleic acid segment used as a marker of bladder cancer or breast cancer, and having a sequence or the complement of a sequence selected from (C), (T) or (U);

(17) an isolated NAM, of 14-100 bases, identical in sequence to a contiguous portion of at least 14 bases of (C), (T) or (U);

(18) an isolated polypeptide encoded by (C), (T) or (U);

(19) an isolated peptide of 10-50 aa, encoded by (C), (T) or (U);

(20) detecting bladder cancer or breast cancer cells by detecting a bladder or breast cancer marker having the sequence (C),

(T) or (U);

(21) treating bladder or breast cancer by:

(a) obtaining a cancer tissue sample;

(b) screening the sample for the expression of a polypeptide encoded by (C), (T) or (U);

(c) providing an antibody against the polypeptide, and

(d) administering the antibody;

(22) treating bladder or breast cancer by:

(a) as in (21a) and (21b);

(b) providing an antisense DNA molecule that encodes a RNA molecule that binds to the polynucleotide;

(c) providing the antisense DNA molecule in the form of a human vector containing regulatory elements for the production of the RNA molecule; and

(d) administering the vector;

(23) a kit for detecting bladder cancer or breast cancer cell, comprising a primer pair for amplifying (C), (T) or (U), ON probe which binds to (C), (T) or (U), or an antibody which binds to the protein encoded by (C), (T) or (U), and

(24) detecting bladder or breast cancer cells by:

(a) providing an antibody that binds to a polypeptide encoded by (C), (T) or (U);

(b) contacting a human tissue sample with the antibody;

(c) separating bound from unbound antibody; and

(d) detecting the bound antibody.

USE - The nucleic acid markers of the invention can be used as markers of prostate cancer, benign prostatic hyperplasia (BPH), bladder cancer or breast cancer, and as targets for therapeutic intervention in prostate cancer, BPH, bladder cancer or breast cancer. The markers may also be used to design specific probes and primers, for the rapid analysis of prostate, bladder or breast biopsy samples. The probes and primers may also be used for in situ hybridization or in situ PCR detection and diagnosis. They may also be used to identify and isolate full length gene sequences form various DNA libraries. Antibodies against the polypeptide products of the markers can be used to treat prostate cancer, bladder cancer or breast cancer. The encoded proteins may be used to detect antibodies. The proteins and antibodies can be used in immunodetection methods for detecting or quantifying the cancers, and for clinical diagnosis of these cancers. The antibodies may also be used for radioimaging to quantify and localize the encoded proteins.

ADVANTAGE - A need exists for the identification of genes which are differentially expressed in prostate, bladder or breast cancer. The present invention meets this need, and provides nucleic acid sequences which can be used in the development of a rapid, inexpensive method to diagnose cancer.

Dwg.0/20

L8 ANSWER 4 OF 17 WPIDS COPYRIGHT 2001 DERWENT INFORMATION LTD
ACCESSION NUMBER: 1999-154991 [14] WPIDS
DOC. NO. NON-CPI: N1999-111853
DOC. NO. CPI: C1999-045913
TITLE: Characterising and identifying disseminated metastatic cancer cells - for diagnosis and prognosis, and for establishing appropriate treatments.
DERWENT CLASS: B04 D16 S03
INVENTOR(S): GIESING, M; AUSTRUP, F; DRIESEL, G; EDER, C; FEIFEL, N; HOLEWA, B; SUCHY, B; UCIECHOWSKI, P
PATENT ASSIGNEE(S): (GIES-I) GIESING M
COUNTRY COUNT: 22
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
DE 19736691	A1	19990225	(199914)*		48
WO 9910528	A1	19990304	(199916)	GE	
RW: AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE					
W: CA JP US					
EP 1007740	A1	20000614	(200033)	GE	
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE					

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
DE 19736691	A1	DE 1997-19736691	19970822
WO 9910528	A1	WO 1998-EP5360	19980824
EP 1007740	A1	EP 1998-946419	19980824
		WO 1998-EP5360	19980824

FILING DETAILS:

PATENT NO	KIND		PATENT NO
EP 1007740	A1	Based on	WO 9910528

PRIORITY APPLN. INFO: DE 1997-19736691 19970822
AN 1999-154991 [14] WPIDS
AB DE 19736691 A UPAB: 19990416
NOVELTY - Characterising and identifying disseminated and metastatic cancer cells using RNA and DNA comprises examining a body fluid sample for at least one **cancer-specific gene** (I) and at least one cancer-associated gene (II).
DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for

agents used in the new process, preferably in test and/or diagnostic kits.

USE - Specifically applied to carcinoma of the **breast**, ovary, lung, pancreas, colon, rectum, prostate and liver; sarcoma; melanoma; non-Hodgkin lymphoma and chronic myeloid leukemia, particularly without lymph node involvement. It is used for diagnosis and prognosis (particularly as regards metastasis or sensitivity to the patient's immune defense system), and as an aid to establish suitable treatments (particularly where characterisation includes identifying resistance genes). It may also be used to monitor treatment or progress of the disease.

ADVANTAGE - The method provides a reliable and accurate assessment of the different forms of cancer (and associated risks) in individual patients. It may be done at any stage in the disease and combined with other standard diagnostic procedures.

Dwg.0/0

L8 ANSWER 5 OF 17 MEDLINE DUPLICATE 1

ACCESSION NUMBER: 2000012325 MEDLINE

DOCUMENT NUMBER: 20012325 PubMed ID: 10546834

TITLE: Search for novel proteins involved in the development of chemoresistance in colorectal cancer and fibrosarcoma cells in vitro using two-dimensional electrophoresis, mass spectrometry and microsequencing.

AUTHOR: Sinha P; Hutter G; Kottgen E; Dietel M; Schadendorf D; Lage H

CORPORATE SOURCE: Institut fur Laboratoriums-medizin und Pathobiochemie, Universitatsklinikum Charite, Medizinische Fakultat der Humboldt Universitat zu Berlin, Germany.. pranav.sinha@charite.de

SOURCE: ELECTROPHORESIS, (1999 Oct) 20 (14) 2961-9. Journal code: ELE; 8204476. ISSN: 0173-0835.

PUB. COUNTRY: GERMANY: Germany, Federal Republic of Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199911

ED Entered STN: 20000111
Last Updated on STN: 20000111
Entered Medline: 19991122

AB In search of novel mechanisms that may lead to the development of chemoresistance of malignant tumors of the large bowel we used two-dimensional electrophoresis to identify proteins that were overexpressed in colorectal and fibrosarcoma cell lines that were resistant towards mitoxantrone. This cytostatic drug is known to lead to atypical multidrug resistance, i.e., the classical mechanism of multidrug resistance (MDR) accompanied by the overexpression of

P-glycoprotein (P-gp) is ineffective. Using mass spectrometry and microsequencing we found adenine phosphoribosyl transferase and **breast cancer specific gene** 1 (BCSG1) overexpressed in the resistant colorectal tumor cell line. In the chemoresistant fibrosarcoma cell line we found two proteins that were overexpressed. One was identified as Rho-guanine dinucleotide phosphate (Rho-GDP) dissociation inhibitor and the other had sequence homologies with yeast protein yer-7. The putative role of these proteins is discussed.

L8 ANSWER 6 OF 17 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 1999137511 MEDLINE
DOCUMENT NUMBER: 99137511 PubMed ID: 9973226
TITLE: Stimulation of **breast** cancer invasion and metastasis by synuclein gamma.
AUTHOR: Jia T; Liu Y E; Liu J; Shi Y E
CORPORATE SOURCE: Department of Pediatrics, Long Island Jewish Medical Center, The Long Island Campus for the Albert Einstein College of Medicine, New Hyde Park, New York 11040, USA.
SOURCE: CANCER RESEARCH, (1999 Feb 1) 59 (3) 742-7.
Journal code: CNF; 2984705R. ISSN: 0008-5472.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199902
ED Entered STN: 19990311
Last Updated on STN: 19990311
Entered Medline: 19990225

AB We recently identified and cloned novel **breast cancer-specific gene** BCSG1 by direct differential cDNA sequencing. BCSG1 has a great sequence homology with the Alzheimer's disease related neural protein synuclein (SNC); thus, it was also named SNC-gamma. Overexpression of SNC-gamma in **breast** cancer cells leads to a significant increase in motility and invasiveness in vitro and a profound augmentation of metastasis in vivo. Our data suggest that this member of the neural protein SNCs might have important functions outside the central nervous system and may play a role in **breast** cancer progression.

L8 ANSWER 7 OF 17 WPIDS COPYRIGHT 2001 DERWENT INFORMATION LTD
ACCESSION NUMBER: 1998-446811 [38] WPIDS
DOC. NO. CPI: C1998-135491
TITLE: New isolated human **breast cancer specific gene** - used to develop products for the diagnosis, clinical management and

treatment of **breast** cancer and metastases.

DERWENT CLASS: B04 D16
INVENTOR(S): JI, H; ROSEN, C A
PATENT ASSIGNEE(S): (HUMA-N) HUMAN GENOME SCI INC
COUNTRY COUNT: 82
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9833915	A1	19980806	(199838)*	EN	72
RW: AT BE CH DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG ZW					
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH GM GW HU ID IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZW					
AU 9862572	A	19980825	(199903)		
EP 1015582	A1	20000705	(200035)	EN	
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE					

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 9833915	A1	WO 1998-US1804	19980203
AU 9862572	A	AU 1998-62572	19980203
EP 1015582	A1	EP 1998-904776	19980203
		WO 1998-US1804	19980203

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9862572	A	Based on	WO 9833915
EP 1015582	A1	Based on	WO 9833915

PRIORITY APPLN. INFO: US 1997-37080 19970203
AN 1998-446811 [38] WPIDS
AB WO 9833915 A UPAB: 19980923
An isolated nucleic acid molecule (NAM) comprises a polynucleotide (PN) having a nucleotide sequence (NS) at least 95% identical to a NS selected from: (a) a NS encoding a polypeptide comprising amino acids (aa) 1/2-127 of 127 aa sequence given in the specification); (b) a NS encoding a polypeptide having an amino acid sequence encoded by the cDNA clones contained in ATCC 97175 or 97856; and (c) a NS complementary to any of the NSs in (a) or (b).

Also claimed are: (1) an isolated NAM comprising a PN which

encodes an aa sequence of an epitope-bearing portion of an **breast cancer specific gene** (BCSG1) polypeptide having an aa sequence as in (a)-(c) above; (2) an isolated NAM comprising a PN having a sequence selected from: (a) a NS of a fragment of a sequence shown where the fragment comprises at least 50 contiguous nucleotides of a sequence shown; and (b) a NS complementary to a NS as in (a); (4) a recombinant vector comprising a NAM as in (a)-(c) above; (5) a recombinant host cell comprising a vector as in (4); (6) an isolated BCSG1 polypeptide having an aa sequence at least 95% identical to a sequence encoded by the NSs as in (a)-(c) above or an epitope-bearing portion of any one of the polypeptides of (a)-(c); (7) a NAM that encodes an epitope bearing portion of a polypeptide as in (a)-(c) above; (8) an isolated polypeptide comprising an epitope-bearing portion of the BCSG1 protein, where the portion is selected from a polypeptide comprising aa residues from 94-107 in a sequence shown and a polypeptide comprising aa residues from 120-127 of the 127b aa polypeptide; (9) an isolated antibody that binds specifically to a BCSG1 polypeptide as in (8); (10) an isolated NAM comprising a PN encoding a BCSG1 polypeptide where, except for 1 to 50 conservative aa substitutions, the polypeptide has a sequence selected from (a)-(c) as above.

USE - The NAMs encode a novel human **breast cancer specific gene** 1 (BCSG1). The BCSG1 is a **breast** cancer marker that is overexpressed in advanced infiltrating **breast** cancer cells. The lack of expression of BCSG1 in normal or benign **breast** epithelial cells and a weak expression in low grade in situ carcinomas suggest that overexpression of BCSG1 indicates **breast** cancer malignant progression. The products can be used for the detection of **breast** cancer cells or **breast** cancer metastasis. They can also be used in the clinical management and treatment of **breast** cancer.

Dwg.0/4

L8 ANSWER 8 OF 17 BIOSIS COPYRIGHT 2001 BIOSIS

ACCESSION NUMBER: 1998:198286 BIOSIS
DOCUMENT NUMBER: PREV199800198286
TITLE: Stimulation of **breast** cancer growth and invasion by a novel **breast cancer specific gene** BCSG1.
AUTHOR(S): Jia, Tongli; Liu, Yiliang E.; Shi, Y. Eric
CORPORATE SOURCE: Long Island Jewish Med. Center, Albert Einstein Coll. Med., New Hyde Park, NY 11040 USA
SOURCE: Proceedings of the American Association for Cancer Research Annual Meeting, (March, 1998) Vol. 39, pp. 649.
Meeting Info.: 89th Annual Meeting of the American Association for Cancer Research New Orleans,

Louisiana, USA March 28-April 1, 1998 American Association for Cancer Research
. ISSN: 0197-016X.
DOCUMENT TYPE: Conference
LANGUAGE: English

L8 ANSWER 9 OF 17 CANCERLIT
ACCESSION NUMBER: 1998639804 CANCERLIT
DOCUMENT NUMBER: 98639804
TITLE: Expression of **breast cancer specific gene** 1 (BCSG1) is suppressed by oncostatin M in **breast** cancer cells (Meeting abstract).
AUTHOR: Anonymous
CORPORATE SOURCE: Mountain States Medical Research Institute and Department of Veterans Affairs Medical Center, Boise, ID 83702.
SOURCE: Proc Annu Meet Am Assoc Cancer Res, (1997). Vol. 38, pp. A2804.
ISSN: 0197-016X.
DOCUMENT TYPE: (MEETING ABSTRACTS)
FILE SEGMENT: ICDB
LANGUAGE: English
ENTRY MONTH: 199808

AB Recently, a novel **breast cancer specific gene**, designated BCSG1, was isolated from differential cDNA sequences derived from **breast** tumors and was partially characterized. We have examined the expression of BCSG1 in several **breast** cancer cell lines. H3922, a cell line derived from infiltrating ductal carcinoma, express the highest level of BCSG1 mRNA among the cell lines that were tested. Since oncostatin M (OM) has an inhibitory effect on H3922 cell growth, we tested whether OM regulates BCSG1 gene expression and found a dramatic suppression of BCSG1 mRNA by OM. A kinetic study showed that treatment of H3922 cells with OM initiated an immediate decrease of BCSG1 mRNA as early as 30 min. By 8 h, the majority of BCSG1 mRNA was gone, and by 24 h, the mRNA was completely undetectable. The effect of OM on BCSG1 was dose-dependent with maximal suppression at concentrations of 1-5 ng/mL. A study of BCSG1 mRNA stability revealed that OM treatment did not alter the mRNA half-life. This suggests that the inhibitory effect of OM on BCSG1 gene expression is at the transcriptional level. In addition to OM, other cytokines and growth factors also modulate the expression of BCSG1. Treatment of H3922 cells with TGFbeta for 24 h increased the mRNA level of BCSG1 up to twofold. However, incubation of cells with EGF and TNFalpha for 24 h decreased the level of BCSG1 mRNA to 50-60% of that in control cells. At present, it is not clear how BCSG1 expression is related to **breast** tumor growth and progression.

L8 ANSWER 10 OF 17 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 97178957 MEDLINE
DOCUMENT NUMBER: 97178957 PubMed ID: 9044857
TITLE: Identification of a **breast cancer -specific gene**, BCSG1, by direct differential cDNA sequencing.
AUTHOR: Ji H; Liu Y E; Jia T; Wang M; Liu J; Xiao G; Joseph B K; Rosen C; Shi Y E
CORPORATE SOURCE: Human Genome Sciences, Inc., Rockville, Maryland 20850-3338, USA.
SOURCE: CANCER RESEARCH, (1997 Feb 15) 57 (4) 759-64.
Journal code: CNF; 2984705R. ISSN: 0008-5472.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AF010126
ENTRY MONTH: 199703
ED Entered STN: 19970313
Last Updated on STN: 20000303
Entered Medline: 19970305

AB A high-throughput direct-differential cDNA sequencing approach was employed to identify genes differentially expressed in normal **breast** as compared with **breast** cancer. Approximately 6000 expressed sequence tags (ESTs) from cDNA libraries of normal **breast** and **breast** carcinoma were selected randomly and subjected to EST-sequencing analysis. The relative expression levels of more than 2000 unique EST groups were quantitatively compared in normal versus cancerous **breast**. Of many putative differentially expressed genes, a **breast cancer-specific gene**, BCSGC1, which was expressed in high abundance in a **breast** cancer cDNA library but scarcely in a normal **breast** cDNA library, was identified as a putative **breast** cancer marker. In situ hybridization analysis demonstrated stage-specific BCSG1 expression as follows: BCSG1 was undetectable in normal or benign **breast** lesions, showed partial expression in ductal carcinoma in situ, but was expressed at an extremely high level in advanced infiltrating **breast** cancer. The predicted amino acid sequence of BCSG1 gene has a significant sequence homology to the non-amyloid beta protein fragment of the Alzheimer's disease amyloid protein. BCSG1 overexpression may indicate **breast** cancer malignant progression from benign **breast** or in situ carcinoma to the highly infiltrating carcinoma.

L8 ANSWER 11 OF 17 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 1997:233002 BIOSIS

DOCUMENT NUMBER: PREV199799532205
TITLE: Expression of **breast** specific gene 1 (BCSG1) is suppressed by oncostatin M in **breast** cancer cells.
AUTHOR(S): Liu, J. (1); Zhang, Y. L.; Jia, T.; Spence, M. J.; Shi, E. Y.
CORPORATE SOURCE: (1) Mountain States Med. Res. Inst., Dep. Veterans Affairs Med. Cent., Boise, ID 83702 USA
SOURCE: Proceedings of the American Association for Cancer Research Annual Meeting, (1997) Vol. 38, No. 0, pp. 419.
Meeting Info.: Eighty-eighth Annual Meeting of the American Association for Cancer Research San Diego, California, USA April 12-16, 1997
ISSN: 0197-016X.
DOCUMENT TYPE: Conference; Abstract
LANGUAGE: English

L8 ANSWER 12 OF 17 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 97025774 EMBASE
DOCUMENT NUMBER: 1997025774
TITLE: Approaches to enhance cancer radiotherapy employing gene transfer methods.
AUTHOR: Buchsbaum D.J.; Raben D.; Stackhouse M.A.; Khazaeli M.B.; Rogers B.E.; Rosenfeld M.E.; Liu T.; Curiel D.T.
CORPORATE SOURCE: D.T. Curiel, University of Alabama at Birmingham, Gene Therapy Program, 1824 6th Avenue South, Birmingham, AL 35294-3300, United States
SOURCE: Gene Therapy, (1996) 3/12 (1042-1068).
Refs: 308
ISSN: 0969-7128 CODEN: GETHEC
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 014 Radiology
016 Cancer
022 Human Genetics
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
AB This review presents an overview and discussion of the potential synergistic strategies of radiation therapy and gene transfer for treating neoplastic disease. Topics discussed include radiation-inducible promoters coupled to genes which produce proteins that are cytotoxic or enhance radiosensitivity, employment of molecular chemotherapy approaches in conjunction with radiation therapy, and genetic induction of radiosensitization through modification of DNA repair, signal transduction, and cell cycle

control genes. Additional topics discussed relate to gene transfer augmentation of radioimmunotherapy of **cancer**. **Specifically**, **gene** transfer methods to genetically induce tumor cells to express enhanced levels of cell surface antigens and receptors to increase radiolabeled antibody and peptide targeting and thus increase their therapeutic effect, selection of radionuclides for therapeutic ligand labeling, and computer simulation of genetic tumor-specific delivery of radioabeled ligands are proposed.

L8 ANSWER 13 OF 17 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 95129061 MEDLINE
DOCUMENT NUMBER: 95129061 PubMed ID: 7828152
TITLE: In vitro karyotype evolution and cytogenetic instability in the non-tumorigenic human **breast** epithelial cell line HMT-3522.
AUTHOR: Nielsen K V; Madsen M W; Briand P
CORPORATE SOURCE: Department of Gynecology and Obstetrics, Rigshospitalet, Copenhagen, Denmark.
SOURCE: CANCER GENETICS AND CYTOGENETICS, (1994 Dec) 78 (2) 189-99.
Journal code: CMT; 7909240. ISSN: 0165-4608.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199502
ED Entered STN: 19950307
Last Updated on STN: 19950307
Entered Medline: 19950217

AB The "spontaneously" immortalized cell line HMT-3522, derived from a fibrocystic **breast** lesion, is used as a model for premalignant **breast** epithelium. During 205 passages the cytogenetic evolution was followed. The results were compared with our earlier results on oncogene expression and growth factor requirements. During in vitro growth, gain and loss of markers, loss of normal chromosomes, and duplication of the chromosome complement could be demonstrated. The variability increased during in vitro growth. This variability, probably created randomly, leads to cells with different growth capacities, from which sidelines may be selected and become stemlines. The karyotypic evolution (including polyploidization) demonstrated here may be a result of genetic instability and heterogeneity. Although tumorigenicity was not achieved, either due to lack of **cancer-specific** **gene** alterations or to lack of proper selection pressure, the results suggest an ongoing process towards malignancy.

L8 ANSWER 14 OF 17 WPIDS COPYRIGHT 2001 DERWENT INFORMATION LTD

ACCESSION NUMBER: 1984-232588 [38] WPIDS
DOC. NO. CPI: C1984-098155
TITLE: Activating gallium-loaded zeolite hydrocarbon aromatisation catalysts - by steaming and hydrogen treatment.
DERWENT CLASS: E14 H04 J04
INVENTOR(S): GANE, B R; HOWARD, P
PATENT ASSIGNEE(S): (BRPE) BRITISH PETROLEUM CO PLC
COUNTRY COUNT: 11
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
EP 119027	A	19840919	(198438)*	EN	16
R: BE DE FR GB IT NL SE					
AU 8425132	A	19840913	(198444)		
JP 59169539	A	19840925	(198444)		
US 4520118	A	19850528	(198524)		
CA 1203226	A	19860415	(198619)		
EP 119027	B	19870916	(198737)	EN	
R: BE DE FR GB IT NL SE					
DE 3466182	G	19871022	(198743)		
JP 04043701	B	19920717	(199233)		8

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
EP 119027	A	EP 1984-301191	19840223
JP 59169539	A	JP 1984-44779	19840308
US 4520118	A	US 1984-580673	19840216
JP 04043701	B	JP 1984-44779	19840308

FILING DETAILS:

PATENT NO	KIND		PATENT NO
JP 04043701	B	Based on	JP 59169539

PRIORITY APPLN. INFO: GB 1983-6531 19830309
AN 1984-232588 [38] WPIDS
AB EP 119027 A UPAB: 19930925
Activation is carried out by treating the zeolite, either before or after the loading with Ga, with steam and H2, concurrently or separately, both stages being carried out at elevated temps. while the Ga-loading, steaming and H2-treating steps may be carried out in any desired sequence, the pref. order is steaming, followed by Ga loading, followed by H2 treatment.

USE/ADVANTAGE - Freshly produced catalysts may be activated, or the steaming and H2 treatment may be applied in the regeneration of (partially) deactivated Ga-loaded zeolite hydrocarbon conversion catalysts. The activated catalysts provide higher conversions and selectivities to aromatics and reduced formation of polynuclear aromatics in the processing of hydrocarbon feedstocks.
0/0

ABEQ EP 119027 B UPAB: 19930925
A process for activating an aluminosilicate zeolite loaded with a gallium cpd as catalyst said process comprising bringing into contact the zeolite, before or after loading thereof with gallium, with steam and concurrently or separately with hydrogen, both stages being carried out at an elevated temp.

ABEQ US 4520118 A UPAB: 19930925
Aluminosilicate zeolite loaded with a Ga-cpd. as catalyst, is activated by contacting zeolite (before or after Ga loading) with steam, and concurrently or separately with H2, each at elevated temp..

Pref. zeolites are aluminosilicates of silica:alumina ratio more than 5:1. Zeolite is subjected to sequence of treatments comprising calcination (C), steaming (S), loading with Ga-cpd. (G) and binding (B) or simultaneous calcination and steaming C(S), in sequence:(a) C(S)GB, (b) C(S)BG, (c) CSGB, (d) CSBG, (e) CGSB, (f) CGBS, (g) CBGS, (h) CBSG, (i) BC(S)G, (j)**BCSG**, or (k) BCGS.

ADVANTAGE - High conversions of hydrocarbon feeds with high selectivity to aromatics are obtd..

L8 ANSWER 15 OF 17 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 5
ACCESSION NUMBER: 1982:183691 BIOSIS
DOCUMENT NUMBER: BA73:43675
TITLE: A CELL SURFACE GLYCO PROTEIN VIRUS INHIBITOR THAT IS NOT INTERFERON.
AUTHOR(S): JOHNSON T C; KINDERS R J; MCGEE J E
CORPORATE SOURCE: SECT. VIROL. ONCOL., DIV. BIOL., KANSAS STATE UNIV., MANHATTAN, KANSAS 66506.
SOURCE: BIOCHEM BIOPHYS RES COMMUN, (1981) 102 (1), 328-334. CODEN: BBRCA9. ISSN: 0006-291X.
FILE SEGMENT: BA; OLD
LANGUAGE: English

AB The possible relationship between a newly isolated glycoprotein virus inhibitor and interferon was assessed. Comparisons of the cell surface glycopeptide, obtained from mouse cerebral cortex, and interferon included antiviral activity, radioumminoassays and the ability of antibodies raised against the brain cell surface glycoprotein (**BCSG**) and against mouse L cell interferon to precipitate the biological activity. **BCSG** was able to inhibit [vesicular stomatitis] virus replication, but only in a

transient fashion. Although anti-**BCSG** precipitated a major portion of the radiolabeled inhibitor in a double antibody assay, anti-mouse interferon did not. Over 90% of the inhibitory activity was removed with anti-**BCSG** and Staphylococcus protein A; anti-mouse interferon removed little or none of the activity under similar reaction conditions. Other properties of the **BCSG** that distinguish it from interferon are presented.

L8 ANSWER 16 OF 17 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 6
ACCESSION NUMBER: 1982:209554 BIOSIS
DOCUMENT NUMBER: BA73:69538
TITLE: GLYCO PEPTIDES PREPARED FROM MOUSE CEREBRUM INHIBIT PROTEIN SYNTHESIS AND CELL DIVISION IN BABY HAMSTER KIDNEY CELLS BUT NOT IN THEIR POLYOMA VIRUS TRANSFORMED ANALOGS.
AUTHOR(S): KINDERS R J; JOHNSON T C
CORPORATE SOURCE: SECTION OF VIROLOGY AND ONCOLOGY, DIVISION OF BIOLOGY, KANSAS STATE UNIVERSITY, MANHATTAN, KS 66506, USA.
SOURCE: EXP CELL RES, (1981) 136 (1), 31-42.
CODEN: ECREAL. ISSN: 0014-4827.
FILE SEGMENT: BA; OLD
LANGUAGE: English
AB Glycopeptides isolated from mouse cerebral cortex cell surfaces (**BCSG**) inhibited cell growth and protein synthesis in baby hamster kidney (BHK)-21 cells, whereas polyoma virus-transformed BHK-21 cells (pyBHK-21) were refractory to the inhibitory activity of the glycopeptides. Growth inhibition was reversible and non-lethal to BHK-21 cells. Despite the difference in sensitivity to the action of the glycopeptides, both cell lines could bind the inhibitor in a saturable fashion and in similar quantities. After trypsinization BHK-21 cells appeared refractory to the inhibitor whereas pyBHK-21 cells became sensitive. The data suggested the presence of a receptor for **BCSG** on the cell surface of both cell lines. Incubating **BCSG** with conditioned medium from pyBHK-21 cells resulted in loss of the glycopeptide's inhibitory activity. Medium conditioned by BHK-21 cells had no effect on the inhibitory activity of **BCSG**. It is hypothesized that the refractoriness of pyBHK-21 cells to **BCSG** is related to their autonomous growth characteristics and failure to respond to topo-inhibitory growth control. **BCSG** may be a naturally occurring growth regulator whose function can be explored by use of the BHK-21/pyBHK-21 model system.

L8 ANSWER 17 OF 17 MEDLINE DUPLICATE 7
ACCESSION NUMBER: 80227767 MEDLINE
DOCUMENT NUMBER: 80227767 PubMed ID: 6248520
TITLE: Glycopeptides from brain inhibit rates of polypeptide

chain elongation.
AUTHOR: Kinders R J; Hughes J V; Johnson T C
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1980 Jul 10) 255 (13) 6368-72.
Journal code: HIV; 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198009
ED Entered STN: 19900315
Last Updated on STN: 19900315
Entered Medline: 19800928

AB In previous reports, we have identified cell-surface glycopeptides from mouse cerebrum (**BCSG**) that inhibited protein synthesis and mitosis in several cell types. When baby hamster kidney (BHK)-21 cells were infected with vesicular stomatitis virus (a negative strand RNA virus), **BCSG** extensively inhibited viral protein synthesis. This inhibition was effective against both protein and glycoprotein synthesis and was independent of amino acid uptake by infected cells, synthesis of viral RNA, and degradation of viral proteins. Analysis of polyribosome profiles in uninfected BHK-21 cells indicated that the degree of cellular protein synthesis inhibition could not be attributed to activation of RNase or solely to a disruption of chain initiation. When added directly to a cell-free protein-synthesizing system derived from BHK-21 cells, **BCSG** was ineffective, but if the inhibitory material was first allowed to react with cells, cell-free protein synthesis was substantially reduced. When **BCSG** were reacted with cells for 5 min at 0 degrees C, the cells tested, BHK-21 (a **BCSG** -sensitive line) and murine fibrosarcoma 2237 (a **BCSG** -insensitive line), both effectively adsorbed the inhibitor from the medium.

FILE 'CAPLUS' ENTERED AT 12:01:07 ON 26 APR 2001
L9 9 SEA ABB=ON PLU=ON (L1 OR L2 OR CSG(S)CANCER(W)SPECIF?) AND METAST?
L10 4 SEA ABB=ON PLU=ON L9 NOT (L4 OR L5)

L10 ANSWER 1 OF 4 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 2000:277882 CAPLUS
DOCUMENT NUMBER: 132:305474
TITLE: Determining expression of **cancer-specific genes** in diagnosing, monitoring, staging, imaging and treating prostate cancer
INVENTOR(S): Salceda, Susana; Recipon, Herve; Cafferkey, Robert

PATENT ASSIGNEE(S): Diadexus Llc, USA
SOURCE: PCT Int. Appl., 74 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000023111	A1	20000427	WO 1999-US24331	19991019

W: CA, JP, US
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE
PRIORITY APPLN. INFO.: US 1998-104737 P 19981019
AB The present invention provides new methods for detecting, diagnosing, monitoring, staging, prognosticating, imaging and treating prostate cancer that involve the detn. of expression of **cancer-specific genes**.
REFERENCE COUNT: 3
REFERENCE(S): (1) Bussemakers; Cancer Research 1999, V59(23), P5975 CAPLUS
(2) Cho-Chung; Current Opinion in Therapeutic Patents 1993, V3(12), P1737
(3) Olsson; Urologic Clinics of North America 1997, V24(2), P367 MEDLINE

L10 ANSWER 2 OF 4 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 2000:277879 CAPLUS
DOCUMENT NUMBER: 132:305473
TITLE: **Cancer-specific gene** expression monitoring for diagnosing, monitoring, staging, imaging and treating prostate cancer
INVENTOR(S): Ali, Shujath M.; Sun, Yongming; Salceda, Susana; Recipon, Herve; Cafferkey, Robert
PATENT ASSIGNEE(S): Diadexus Llc, USA
SOURCE: PCT Int. Appl., 35 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000023108	A1	20000427	WO 1999-US23764	19991018

W: CA, JP, US

RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE
PRIORITY APPLN. INFO.: US 1998-104741 P 19981019
AB The present invention provides a new method involving measuring levels of **cancer-specific gene** expression for detecting, diagnosing, monitoring, staging, prognosticating, imaging and treating prostate cancer.
REFERENCE COUNT: 3
REFERENCE(S): (1) Bussemakers; Cancer Research 1999, V59(23), P5975 CAPLUS
(2) Cho-Chung; Current Opinion in Therapeutic Patents 1993, V3(12), P1737
(3) Olsson; Urologic Clinics of North America 1997, V24(2), P367 MEDLINE

L10 ANSWER 3 OF 4 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 2000:241570 CAPLUS
DOCUMENT NUMBER: 132:292266
TITLE: A novel method of diagnosing, monitoring, staging, imaging and treating gastrointestinal cancers using **cancer specific gene** CC2
INVENTOR(S): Macina, Roberto A.
PATENT ASSIGNEE(S): Diadexus LLC, USA
SOURCE: PCT Int. Appl., 33 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000020640	A1	20000413	WO 1999-US22725	19990930

W: CA, JP, US
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE
PRIORITY APPLN. INFO.: US 1998-102879 P 19981002
AB A diagnostic and **metastatic** marker CC2 (colon cancer-specific 2) useful not only for colon cancer but also for cancer of the stomach and small intestine is provided. Also provided cDNA and protein sequences of CC2, and antibodies against CC2. CC2 mRNA is highly expressed in normal tissues from gastrointestinal tract, with a lower level of expression in prostate and testis, and deferentially expressed in gastrointestinal cancers. A method for detecting, diagnosing, monitoring, staging, prognosticating, imaging and treating gastrointestinal cancers including small intestine, colon and stomach cancer via CC2 is

provided.
REFERENCE COUNT: 3
REFERENCE(S): (1) Human Genome Sciences Inc; WO 9639541 A1 1996 CAPLUS
(2) Incyte Pharmaceuticals Inc; WO 9816640 A1 1998 CAPLUS
(3) Soppet; US 5861494 A 1999 CAPLUS

L10 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1997:105216 CAPLUS
DOCUMENT NUMBER: 126:116609
TITLE: A gene expressed in colon cancers and its gene product and their diagnostic and therapeutic uses
INVENTOR(S): Soppet, Daniel R.; Li, Yi; Dillon, Patrick J.
PATENT ASSIGNEE(S): Human Genome Sciences, Inc., USA; Soppet, Daniel R.; Li, Yi; Dillon, Patrick J.
SOURCE: PCT Int. Appl., 64 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9639541	A1	19961212	WO 1995-US7169	19950606
W: AM, AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, ES, FI, GB, GE, HU, JP, KE, KG, KP, KR, KZ, LK, LT, LU, LV, MD, MG, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SI, SK, TJ, TT, UA, US, UZ, VN				
RW: KE, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 9528180	A1	19961224	AU 1995-28180	19950606
AU 711346	B2	19991014		
EP 833948	A1	19980408	EP 1995-923729	19950606
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE				

PRIORITY APPLN. INFO.: WO 1995-US7169 19950606
AB A gene that is expressed in cancerous colon tissue and that may be used as a diagnostic marker or as a target for treatment of the disease (no data) is described. The gene can also be used as a marker for **metastasis** of the tumor. Antibodies specific to the gene product that may be used to target cancer cells and as part of a colon cancer vaccine are also described. Methods of screening for agonists and antagonists for the polypeptide and therapeutic uses of the antagonists are also disclosed. Expression

of the cloned gene in a baculovirus system is described.

(FILE 'MEDLINE, BIOSIS, EMBASE, WPIDS, CONFSCI, SCISEARCH, JICST-EPLUS, JAPIO, CANCERLIT' ENTERED AT 12:03:12 ON 26 APR 2001)

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L11          16 S L9
L12           5 S L11 NOT L7
L13           5 DUP REM L12 (0 DUPLICATES REMOVED)
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L13 ANSWER 1 OF 5 WPIDS COPYRIGHT 2001 DERWENT INFORMATION LTD

ACCESSION NUMBER: 2000-339531 [29] WPIDS
DOC. NO. NON-CPI: N2000-254921
DOC. NO. CPI: C2000-103001
TITLE: Diagnosing, staging and monitoring the presence and **metastases** of prostate cancer especially useful for treating prostate cancer comprises measuring changes in **cancer specific gene** levels.
DERWENT CLASS: B04 D16 S03
INVENTOR(S): CAFFERKEY, R; RECIPON, H; SALCEDA, S
PATENT ASSIGNEE(S): (DIAD-N) DIADEXUS LLC
COUNTRY COUNT: 21
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2000023111	A1	20000427	(200029)*	EN	74

RW: AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE
W: CA JP US

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000023111	A1	WO 1999-US24331	19991019

PRIORITY APPLN. INFO: US 1998-104737 19981019
AN 2000-339531 [29] WPIDS
AB WO 200023111 A UPAB: 20000617
NOVELTY - A method for diagnosing the presence of prostate cancer in a patient, comprising determining levels of **cancer specific genes** (**CSG**) in cells, tissues or bodily fluids, and comparing the determined levels of **CSG** with levels of **CSG** from a normal human control, is new. A change in determined levels of **CSG** in the patient versus the control is associated with the presence of prostate cancer.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) a method of diagnosing **metastases** of prostate cancer in a patient comprising:

(a) identifying a patient having prostate cancer that is not known to have **metastasized**;

(b) determining CSG levels in a sample of cells, tissues, or bodily fluid from the patient; and

(c) comparing the determined CSG levels with CSG levels of a normal human control, where an increase in CSG levels in the patient versus the control, is associated with a cancer which has **metastasized**;

(2) a method of staging prostate cancer in a patient having prostate cancer, comprising:

(a) identifying a patient having prostate cancer;

(b) determining CSG levels in a sample of cells, tissue, or bodily fluid from the patient; and

(c) comparing determined CSG levels with CSG levels of a normal human control, where an increase in CSG levels in the patient versus the control is associated with a progressing cancer, and a decrease in the CSG levels is associated with a regressing cancer;

(3) a method of monitoring prostate cancer in a patient for the onset of **metastasis** comprising:

(a) identifying a patient having prostate cancer that is not known to have **metastasized**;

(b) periodically determining CSG levels in samples of cells, tissues, or bodily fluid from the patient; and

(c) comparing the CSG levels with CSG levels of CSG of a normal human control, where an increase in any one of the periodically determined CSG levels in the patient versus the control is associated with a cancer which has **metastasized**;

(4) a method of monitoring a change in stage of prostate cancer in a patient comprising:

(a) identifying a patient having prostate cancer;

(b) periodically determining levels of CSG;

(c) comparing the CSG levels with CSG levels of a normal human control, where an increase in any one of the periodically determined CSG levels in the patient versus the control is associated with a progressing cancer, and a decrease is associated with a regressing cancer;

(5) a method of identifying potential therapeutic agents for use in imaging and treating prostate cancer, comprising screening molecules for an ability to bind to CSG, which indicates the molecule is useful in imaging and treating prostate cancer;

(6) an antibody which specifically binds CSG; and

(7) a method of imaging or treating prostate cancer in a patient by administering an antibody of (7).

ACTIVITY - Cytostatic.

MECHANISM OF ACTION - The antibody, conjugated to a cytotoxic agent, binds to **cancer specific genes**,

in vivo.

USE - The method is useful for diagnosing, staging and monitoring the presence and **metastases** of cancer (claimed). The antibodies which specifically binds CSG or fragments of such antibodies can be used in treating prostate cancer, and to detect or image, localization of CSG in a patient in order to diagnose a disease or condition (claimed). The antibodies may also be used in the treatment of diseases characterized by the expression of CSG.

ADVANTAGE - The new method provides an earlier diagnosis for the presence and **metastasis** of prostate cancer, which significantly increase the chances of a cure. It provides a sensitive method for diagnosing, and staging, prostate cancer to determine if the cancer has **metastasized**, and for monitoring the progress or stage of the disease, which has not **metastasized**.

Dwg.0/0

L13 ANSWER 2 OF 5 WPIDS COPYRIGHT 2001 DERWENT INFORMATION LTD

ACCESSION NUMBER: 2000-339528 [29] WPIDS
DOC. NO. NON-CPI: N2000-254918
DOC. NO. CPI: C2000-102998
TITLE: Diagnosing, detecting, staging, monitoring, imaging and treating cancers, especially useful for detecting prostate cancer comprises measuring changes in levels of **cancer specific genes** in cells, tissues and body fluids.
DERWENT CLASS: B04 D16 S03
INVENTOR(S): ALI, S M; CAFFERKEY, R; RECIPON, H; SALCEDA, S; SUN, Y
PATENT ASSIGNEE(S): (DIAD-N) DIADEXUS LLC
COUNTRY COUNT: 21
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2000023108	A1	20000427	(200029)*	EN	33

RW: AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE
W: CA JP US

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000023108	A1	WO 1999-US23764	19991018

PRIORITY APPLN. INFO: US 1998-104741 19981019
AN 2000-339528 [29] WPIDS
AB WO 200023108 A UPAB: 20000617
NOVELTY - A method for diagnosing the presence of prostate cancer, comprising measuring levels of **cancer specific genes (CSG)** in cells, tissues or bodily fluids, and comparing the measured **CSG** levels with levels from a normal human control, where a change in measured **CSG** levels in the patient versus the control is associated with the presence of prostate cancer.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) a method for diagnosing the **metastases** of prostate cancer in a patient comprising:

(a) identifying a patient having prostate cancer that is not known to have **metastasized**;

(b) measuring CSG levels in cells, tissues or bodily fluid of the patient; and

(c) comparing the measured CSG levels with levels of a normal human control, where an increase in measured CSG levels in the patient versus the control is associated with a cancer which has **metastasized**;

(2) a method for staging prostate cancer in a patient having prostate cancer comprising:

(a) identifying a patient having prostate cancer;

(b) measuring CSG levels in cells, tissues or bodily fluid of the patient; and

(c) comparing the measured CSG levels with levels of a normal human control, where an increase in measured CSG levels in the patient versus the control is associated with a cancer which is progressing, and a decrease in the measured CSG levels is associated with a cancer which is regressing or in remission;

(3) a method of monitoring prostate cancer in a patient for the onset of **metastasis** comprising:

(a) identifying a patient having prostate cancer that is known to have **metastasized**;

(b) periodically measuring levels of CSG in samples of cells, tissues or bodily fluid from the patient;

(c) comparing the CSG levels with levels of a normal human control, where an increase in any one of the periodically measured CSG levels in the patient versus the control is associated with a cancer which has **metastasized**;

(4) a method of monitoring a change in stage of prostate cancer in a patient comprising:

(a) identifying a patient having prostate cancer;

(b) periodically measuring levels of CSG in samples of cells, tissues or bodily fluid from the patient;

(c) comparing the CSG levels with levels of a normal human

control, where an increase in any one of the periodically measured CSG levels in the patient versus the control is associated with a progressing cancer, and a decrease is associated with a regressing cancer.

(5) an antibody which specifically binds CSG; and

(6) a method of imaging or treating prostate cancer in a patient, comprising administering the antibody of (6).

ACTIVITY - Cytostatic.

MECHANISM OF ACTION - The antibody, conjugated to a cytotoxic agent, binds to **cancer specific genes**.

USE - The method is useful for diagnosing, detecting, staging, monitoring, and imaging for the presence and **metastases** of prostate cancer. The antibodies which specifically bind to CSG may be used to detect or image localization of CSG in a patient in order to detect or diagnose a disease or condition, and to treat prostate cancer. All claimed.

ADVANTAGE - The new method provides an earlier diagnosis for the presence and **metastasis** of prostate cancer, which significantly increase the chances of cure. It provides a sensitive method for diagnosing and staging of prostate cancer to determine whether or not such cancer has **metastasized**, and for monitoring the progress of the disease, which has not **metastasized** for the onset of **metastasis**.

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L13 ANSWER 3 OF 5 WPIDS COPYRIGHT 2001 DERWENT INFORMATION LTD

ACCESSION NUMBER: 2000-328946 [28] WPIDS

DOC. NO. NON-CPI: N2000-247638

DOC. NO. CPI: C2000-099678

TITLE: Detecting, diagnosing and monitoring gastrointestinal cancers comprises measuring the levels of **cancer specific gene**/protein 2 (CC2) in tissues or bodily fluids.

DERWENT CLASS: B04 D16 S03

INVENTOR(S): MACINA, R A

PATENT ASSIGNEE(S): (DIAD-N) DIADEXUS LLC

COUNTRY COUNT: 21

PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2000020640	A1	20000413	(200028)*	EN	33

RW: AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE

W: CA JP US

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000020640 A1		WO 1999-US22725	19990930

PRIORITY APPLN. INFO: US 1998-102879 19981002
AN 2000-328946 [28] WPIDS
AB WO 200020640 A UPAB: 20000613
NOVELTY - Diagnosing the presence of gastrointestinal cancer (GC), comprising measuring a change in levels of **cancer specific gene**/protein 2 (CC2) in cells, tissues or bodily fluids in a patient compared with CC2 levels in a normal human control, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) diagnosing **metastases** of a GC in a patient, comprising:

(a) identifying a patient having a GC that is not known to have **metastasized**; and

(b) the above new method. where an increase in measured CC2 levels in the patient is associated with a cancer which has **metastasized**;

(2) staging a GC in a patient having a GC, comprising steps (a)-(b) of method of (1), where an increase in CC2 levels in the patient is associated with a cancer which is progressing and a decrease is associated with a cancer which is regressing or in remission;

(3) monitoring a change in the stage of a GC in a patient, comprising step (a) of the method of (1) and:

(a) periodically measuring the level of CC2 in samples of cells, tissues or bodily fluids from the patient; and

(b) as for step (c) of the method of (1), wherein an increase in CC2 levels in the patient is associated with a cancer which has **metastasized**/is progressing and a decrease is associated with a cancer which is regressing or in remission;

(4) an antibody that specifically binds CC2;

(5) imaging a GC cancer in a patient, comprising administering the antibody of (4) (which is preferably labeled with paramagnetic ions or a radioisotope) to the patient; and

(6) a method of treating a GC in a patient, comprising administering the antibody of (5) (which is preferably conjugated to a cytotoxic agent) to the patient.

USE - The methods are used for diagnosing the presence of gastrointestinal cancers such as stomach cancer, cancer of the small intestine, and colon cancer, especially for a gastrointestinal cancer which has not **metastasized**. The methods may also be used for staging and monitoring gastrointestinal cancer. Antibodies which specifically bind to colon specific gene 2 (CC2) can also be

used in vivo in patients suspected of having gastrointestinal cancers, for treatment and imaging (all claimed).

ADVANTAGE - The new methods are sensitive and specific and allow for early diagnosis of gastrointestinal cancer. This means that treatment can commence earlier. Furthermore, the methods are not invasive, unlike prior art surgical procedures.

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L13 ANSWER 4 OF 5 WPIDS COPYRIGHT 2001 DERWENT INFORMATION LTD

ACCESSION NUMBER: 2000-283453 [24] WPIDS
DOC. NO. NON-CPI: N2000-213335
DOC. NO. CPI: C2000-085572
TITLE: Methods for diagnosing, staging, imaging and treating gynecologic and testicular cancers by measuring expression of a **cancer specific gene**.
DERWENT CLASS: B04 D16 S03
INVENTOR(S): ALI, S M; CAFFERKEY, R
PATENT ASSIGNEE(S): (DIAD-N) DIADEXUS LLC
COUNTRY COUNT: 21
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2000016805	A1	20000330	(200024)*	EN	33

RW: AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE
W: CA JP US

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000016805	A1	WO 1999-US21774	19990923

PRIORITY APPLN. INFO: US 1998-101522 19980923
AN 2000-283453 [24] WPIDS
AB WO 200016805 A UPAB: 20000522
NOVELTY - Methods ((I) - (IV)) for diagnosing, staging, imaging and treating gynecologic and testicular cancers by measuring expression of a **cancer specific gene** (**CSG**) (comprising a defined 1081 nucleotide sequence given in the specification), are new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:

(1) a method (I) for diagnosing the presence of a gynecological or testicular cancer in a patient, comprising:

(i) measuring the levels of CSG in cells, tissues or bodily

fluids of the patient; and

(ii) comparing the measured levels of CSG with the levels found in a normal human control (a change in the measured level of CSG is associated with the presence of the cancer);

(2) a method (II) for diagnosing and monitoring **metastases** of a gynecological or testicular cancer, comprising:

(i) identifying a patient suffering from a cancer that is not known to have **metastasized**;

(ii) periodically measuring CSG levels in samples of cells, tissues or fluids from the patient; and

(iii) comparing the measured levels of CSG with the levels found in a normal human control (an increase in the measured level of CSG is associated with the presence of a cancer that has **metastasized**);

(3) a method (III) of staging a gynecological or testicular cancer, comprising:

(i) identifying a patient with the cancer;

(ii) periodically measuring levels of CSG in samples of cells tissues or fluids from the patient; and

(iii) comparing the measured levels of CSG with the levels found in a normal human control (an increase in the measured level of CSG is associated with the progression of the cancer and a decrease in the levels is associated with the remission of the cancer);

(4) an antibody (Ab) against CSG;

(5) a method (IV) of imaging a gynecological or testicular cancer comprising administering Ab; and

(6) a method (V) of treating a gynecological or testicular cancer comprising administering Ab.

USE - (I) - (IV) may used for be diagnosing, staging, imaging and treating gynecologic and testicular cancers.

ADVANTAGE - Early diagnosis of cancers improves the success rate of therapeutic protocols.

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L13 ANSWER 5 OF 5 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 97:307617 SCISEARCH
THE GENUINE ARTICLE: WT711
TITLE: Reverse transcriptase-polymerase chain reaction assays for prostate cancer
AUTHOR: Olsson C A (Reprint); deVries G M; Buttyan R; Katz A E
CORPORATE SOURCE: COLUMBIA UNIV, COLL PHYS & SURG, SQUIER UROL CLIN, ATCHLEY PAVIL, 11TH FLOOR, 161 FT WASHINGTON AVE, NEW YORK, NY 10032 (Reprint); COLUMBIA UNIV, COLL PHYS & SURG, DEPT UROL, NEW YORK, NY
COUNTRY OF AUTHOR: USA

09/721183

SOURCE: UROLOGIC CLINICS OF NORTH AMERICA, (MAY 1997) Vol. 24, No. 2, pp. 367-&.
Publisher: W B SAUNDERS CO, INDEPENDENCE SQUARE WEST CURTIS CENTER, STE 300, PHILADELPHIA, PA 19106-3399.
ISSN: 0094-0143.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: CLIN
LANGUAGE: English
REFERENCE COUNT: 80

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The use of reverse transcriptase-polymerase chain reaction assays (RT-PCR) directed against prostate or prostate **cancer-specific genes** is a controversial topic. This article reviews the field thoroughly to delineate areas where consensus may have been reached versus those areas of continuing controversy. Regardless of the tissue substrate tested (blood, marrow), there is apparent consensus that patients without cancer have negative assays. There is additional consensus supporting that untreated patients with **metastatic** disease have positive assays. At least three groups have shown that RT-PCR assays of prostate-specific antigen mRNA provide unique prognostic information in the patients with clinically localized disease. There is continuing controversy of whether RT-PCR directed against any marker gene is of value in staging prostate cancer.

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